# AURICULAR PREMATURE SYSTOLES: THE DURATION OF THE ELECTRICAL SYSTOLE

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In a recent electrocardiographic investigation (Berliner and Lewithin, 1945) the aberration of the ventricular complex of auricular premature systoles (*APS*)* was studied. Variations in amplitude, duration, direction, and configuration were found and described in detail. In the course of that investigation, a new feature of the *APS*, shortening of the electrical systole (Q–T interval), was discovered. This subject has since been studied by careful measurements which are reported in the present paper.

## Material and Method

We used 100 electrocardiograms of different patients in this study, selected from the records of over 250 patients exhibiting *APS*. The selection was made on the basis of technical qualities of the tracings. We chose only clear records in which the exact end-points of the T waves were easily discernible. Tracings blurred by muscle tremor or technical defects were eliminated; as were such perfect records in which low amplitude of the T waves made accurate measurement of the Q–T interval impossible; and also all those records in which the *APS* were found to occur at the beginning of a tracing or at its very end (with the exception of Fig. 2A). Records with inverted T waves were purposely included.

Of the four leads available in practically all cases, we selected that lead in which the end-points of the T waves were most distinct, and analyzed one *APS* of that lead. The degree of prematurity of this *APS* and the degree of its aberration were determined first, and for this purpose the criteria established in the previous study were again used. Measurements were then made on five successive heart beats, the two beats preceding the *APS*, the *APS* itself, and the two beats following it. All measurements were made with the aid of a magnifying glass. The duration of QRS and RS–T † was measured in each of the five beats. The Q–T intervals were then obtained by adding up the values for QRS and RS–T. In the normal beat just preceding the *APS*, however, RS–T and Q–T could usually not be measured because the P wave of the *APS* was superimposed on its T wave. That was the reason why the beat preceding it was also measured and was chosen as *the* normal for purposes of comparison. QRS, RS–T, and Q–T of the *APS* were compared with the respective intervals of the normal beat and the post-extrasystolic beat. Examples are given in the legend to Fig. 1.

## Results

*QRS duration and Q–T interval.* In 47 of our 100 *APS*, the duration of QRS did not differ from that of the normal beats or the difference was too minute for measurement. In 23, QRS duration was shortened; this shortening never exceeded 0·02 second. In 30 the QRS complexes were prolonged. The prolongation was marked (0·02 second or more) in 15 of these. Marked prolongation was usually associated with other features of aberration, viz.

* *APS* will be used as an abbreviation for auricular premature systole(s) throughout this paper.
† By RS–T interval we mean the interval beginning at the RS–T junction and ending at the end of the T wave.

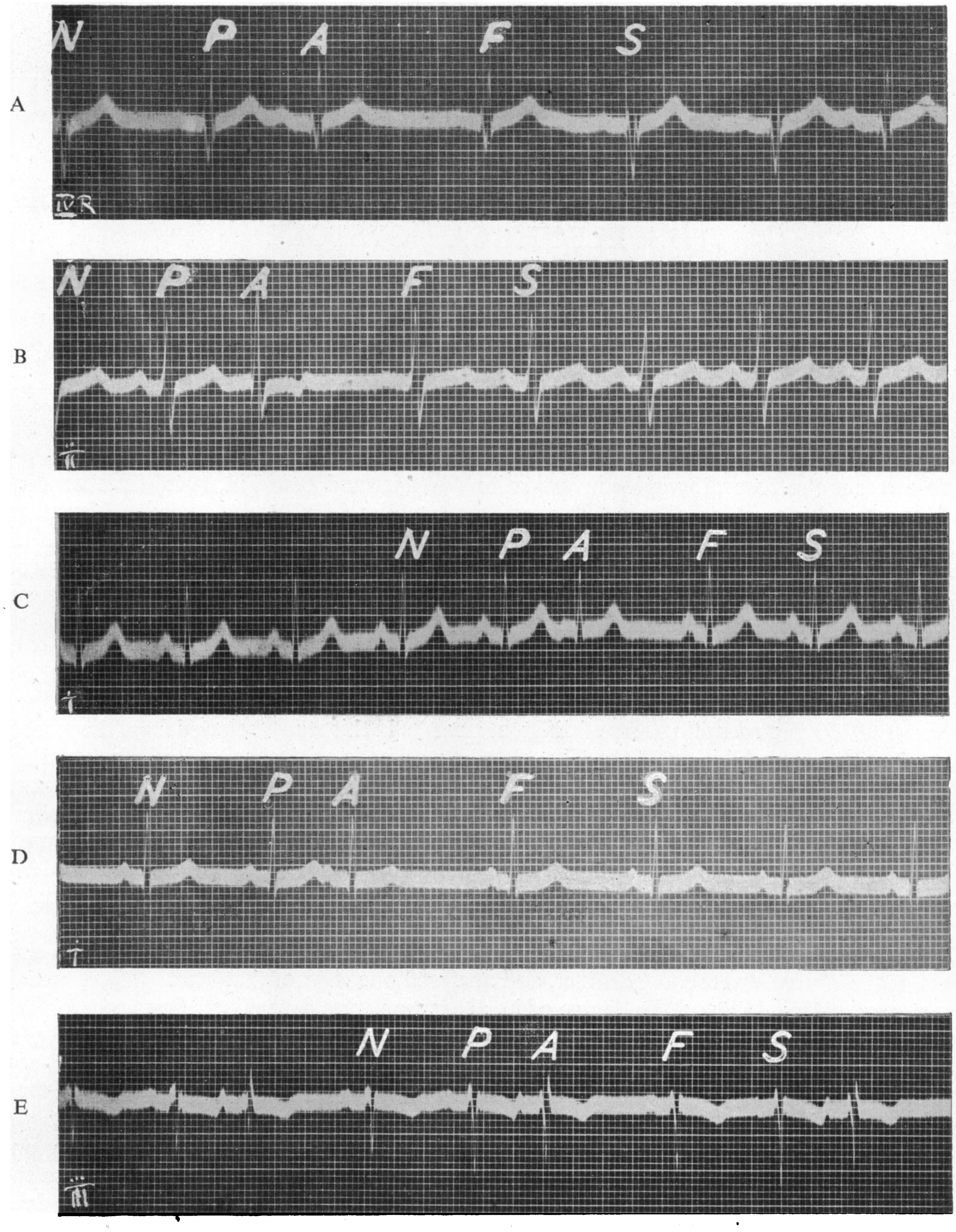



FIG. 1.—Shortening of the electrical systole in auricular premature systoles (*APS*)—five typical examples. QRS duration essentially unchanged, RS–T duration (from the RS–T junction to the end of T) shortened. N, normal beat; P, beat preceding the *APS*; A, *APS*; F, beat following the *APS*; S, subsequent beat. Measurements in seconds:

| (A) | QRS | RS–T | Q–T |
|---|---|---|---|
| N | 0·075 | 0·275 | 0·350 |
| P | 0·075 | 0·275 | 0·350 |
| A | 0·075 | 0·260 | 0·335 |
| F | 0·080 | 0·285 | 0·365 |
| S | 0·080 | 0·280 | 0·360 |

| (B) | QRS | RS–T | Q–T |
|---|---|---|---|
| N | cut off | 0·28 | × |
| P | 0·085 | × | × |
| A | 0·085 | 0·26 | 0·345 |
| F | 0·080 | 0·29 | 0·370 |
| S | 0·095 | 0·28 | 0·375 |

| (C) | QRS | RS–T | Q–T |
|---|---|---|---|
| N | 0·07 | 0·30 | 0·37 |
| P | 0·075 | × | × |
| A | 0·07 | 0·29 | 0·36 |
| F | 0·07 | 0·32 | 0·39 |
| S | 0·07 | 0·30 | 0·37 |

| (D) | QRS | RS–T | Q–T |
|---|---|---|---|
| N | 0·07 | 0·31 | 0·38 |
| P | 0·065 | × | × |
| A | 0·07 | 0·28 | 0·35 |
| F | 0·07 | 0·32 | 0·39 |
| S | 0·065 | 0·32 | 0·385 |

| (E) | QRS | RS–T | Q–T |
|---|---|---|---|
| N | 0·07 | 0·32 | 0·39 |
| P | 0·07 | × | × |
| A | 0·07 | 0·25 | 0·32 |
| F | 0·07 | 0·31 | 0·38 |
| S | 0·07 | × | × |

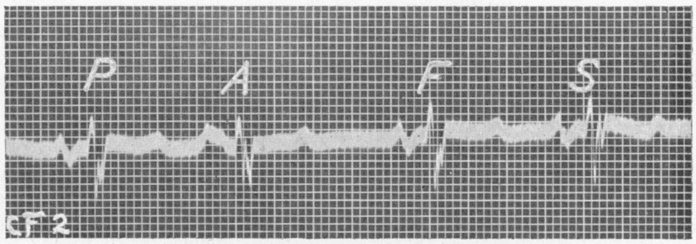
changes in amplitude, configuration, or direction. When we measured the duration of RS–T in the 15 *APS* which showed marked prolongation of QRS, we often found marked shortening of RS–T. So great was the shortening of RS–T in 9 of these 15 cases, that it " compensated " for the increase in QRS duration, making the total Q–T interval of the *APS* equal to the Q–T interval of the normal beats or even shorter. An example will illustrate this point (Fig. 3A, compare A with N).

| | |
|---|---|
| QRS of *APS* (A) prolonged by | 0·05 second |
| RS–T of *APS* (A) shortened by | 0·06 second |
| Q–T interval of *APS* (A) shortened by | 0·01 second |

Our measurements of the QRS duration of *APS* may be summed up by the statement that changes in QRS duration did not significantly affect the Q–T interval of the *APS*.

*Measurements of RS–T.* The Q–T interval of an *APS* is usually shortened. This shortening occurs in the RS–T interval. When the RS–T interval of the *APS* was compared with that of the normal beat (N), it was found shortened in 82 per cent of the cases (Fig. 1). In 12 per cent there was no difference in RS–T duration, and in only 6 per cent was there a slight prolongation (Fig. 2).

A




B

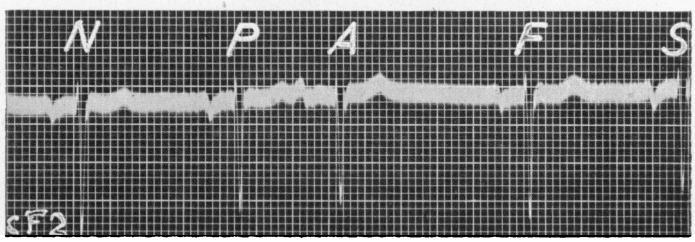



FIG. 2.—Slight lengthening of the electrical systole in auricular premature systoles (*APS*)—two examples of an unusual condition. QRS duration essentially unchanged, RS–T duration (from the RS–T junction to the end of T) lengthened. N, normal beat; P, beat preceding the *APS*; A, *APS*; F, beat following the *APS*; S, subsequent beat. Measurements in seconds.

(A)

| | QRS | RS–T | Q–T |
|---|---|---|---|
| N | | cut off | |
| P | 0·095 | 0·325 | 0·42 |
| A | 0·09 | 0·33 | 0·42 |
| F | 0·095 | 0·33 | 0·425 |
| S | 0·10 | 0·32 | 0·42 |

(B)

| | QRS | RS–T | Q–T |
|---|---|---|---|
| N | 0·07 | 0·26 | 0·33 |
| P | 0·08 | 0·26 | 0·34 |
| A | 0·08 | 0·27 | 0·35 |
| F | 0·07 | 0·29 | 0·36 |
| S | | cut off | |

We were unable to discover what causes the shortening of RS–T in the *APS*. Various possible factors were investigated. The effect of T-wave inversion was easily ruled out. Leads in which all T waves or only the T waves of the *APS* were inverted showed no more shortening than leads in which they were upright. We then studied the effect which the degree of prematurity of an *APS* might have on its RS–T duration. For this purpose, we compared the RS–T intervals of early and late *APS*. Our series included 15 early *APS*; their P waves were superimposed on the ascending limb of the preceding T wave (" location 4 " of the previous paper). Our series also included 23 late *APS* the P waves of which did not touch the preceding T waves (" location 1 " of the previous paper). Shortening of RS–T was no more marked in the early *APS* than in the late ones, and the frequency of RS–T shortening was the same in both groups (Table I). We concluded that the degree of prematurity of an *APS* has no influence on the duration of its RS–T interval.

TABLE I.—COMPARISON OF EARLY AND LATE AURICULAR PREMATURE SYSTOLES (*APS*)

| | Number over of cases | RS–T interval shorter than normal | RS–T interval identical with normal beats | RS–T interval longer than normal | Shortening of RS–T varied |
|---|---|---|---|---|---|
| Early *APS* (location 4) | 15 | 13 | 0 | 2 | From 0·01 to 0·07 second. |
| Late *APS* (location 1) | 23 | 19 | 3 | 1 | From 0·005 to 0·06 second. |

Another possible factor investigated by us was the degree of aberration, but it too was ruled out. *APS* with aberration showed no more shortening of RS–T than *APS* without any aberration. One group of markedly aberrant *APS*, however, formed an exception, viz. *APS* with marked prolongation of their QRS complexes. As mentioned above, in most of these *APS* (9 out of 15 cases) prolongation of QRS was accompanied by an extra marked shortening of RS–T. At times, this shortening of RS–T was so marked that it overbalanced the prolongation of QRS; the duration of Q–T was then shorter than that of the normal beats (Fig. 3A). The highest degrees of RS–T shortening, by as much as 0·06 and 0·07 sec., occurred in

A

B

C

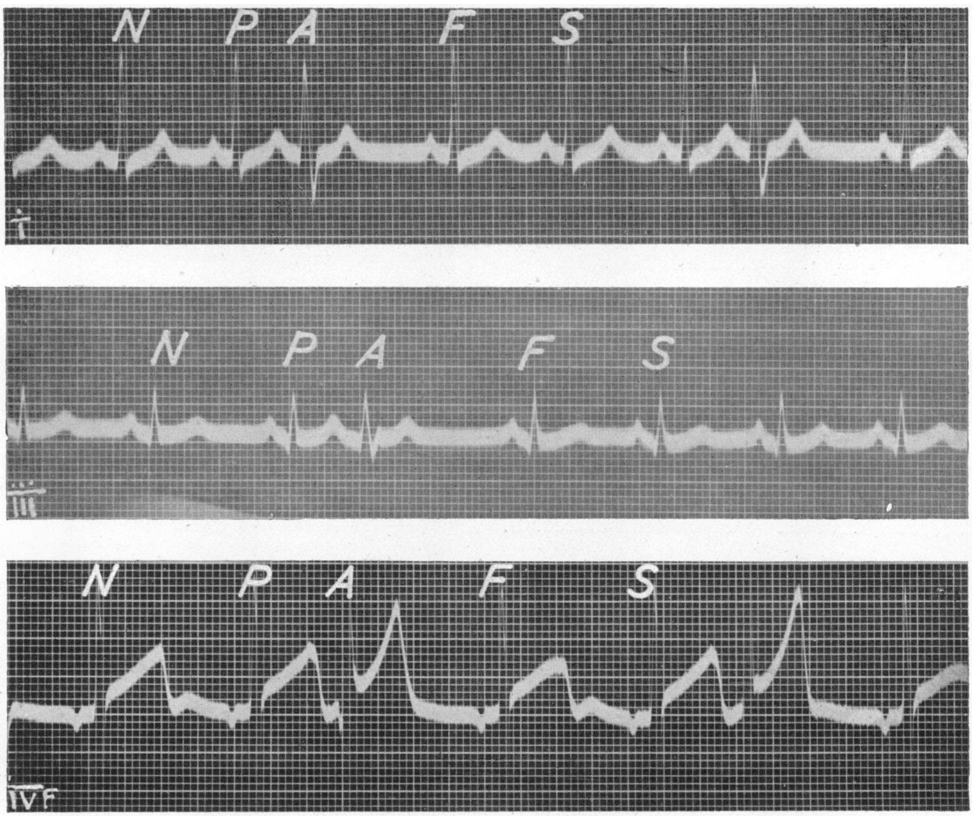



FIG. 3.—Auricular premature systoles (*APS*) showing increased QRS duration but markedly decreased RS–T duration—three examples. N, normal beat; P, beat preceding the *APS*; A, *APS*; F, beat following the *APS*; S, subsequent beat. Measurements in seconds.

| (A) | QRS | RS–T | Q–T |
|---|---|---|---|
| N | 0·08 | 0·31 | 0·39 |
| P | 0·08 | × | × |
| A | 0·13 | 0·25 | 0·38 |
| F | 0·09 | 0·31 | 0·40 |
| S | 0·08 | 0·31 | 0·39 |

| (B) | QRS | RS–T | Q–T |
|---|---|---|---|
| N | 0·06 | 0·28 | 0·34 |
| P | 0·07 | × | × |
| A | 0·12 | 0·23 | 0·35 |
| F | 0·07 | 0·29 | 0·36 |
| S | 0·07 | 0·29 | 0·36 |

| (C) | QRS | RS–T | Q–T |
|---|---|---|---|
| N | 0·065 | 0·37 | 0·435 |
| P | 0·07 | × | × |
| A | 0·10 | 0·34 | 0·44 |
| F | 0·07 | 0·38 | 0·45 |
| S | 0·07 | × | × |

such *APS* with markedly prolonged QRS complexes. This may be more than a coincidence; in fact, it suggests a possible interdependence of QRS and RS–T. There were, on the other hand, 3 cases in which such "compensation" did not take place; both the QRS and the RS–T intervals were prolonged, resulting in marked prolongation of Q–T, up to 0·12 sec. (Fig. 4A and Table II).

TABLE II

AURICULAR PREMATURE SYSTOLES (*APS*) WITH MARKED INCREASE IN QRS DURATION. (Measurements in seconds.)

| | QRS duration increased by | RS–T duration shortened by | RS–T duration lengthened by | Resulting Q–T compared to normal | Illustrated in |
|---|---|---|---|---|---|
| 1 | 0·02 | 0·04 | — | −0·02 | |
| 2 | 0·02 | 0·04 | — | −0·02 | |
| 3 | 0·02 | 0·02 | — | identical | |
| 4 | 0·02 | 0·01 | — | +0·01 | |
| 5 | 0·03 | 0·03 | — | identical | Fig. 3C |
| 6 | 0·03 | 0·03 | — | identical | |
| 7 | 0·03 | 0·02 | — | +0·01 | |
| 8 | 0·03 | 0·01 | — | +0·02 | |
| 9 | 0·03 | — | 0·01 | +0·04 | |
| 10 | 0·04 | 0·04 | — | identical | |
| 11 | 0·04 | — | 0·035 | +0·075 | Fig. 4A |
| 12 | 0·04 | — | 0·08 | +0·12 | Fig. 4B |
| 13 | 0·05 | 0·06 | — | −0·01 | Fig. 3A |
| 14 | 0·05 | 0·05 | — | identical | |
| 15 | 0·06 | 0·05 | — | +0·01 | Fig. 3B |

A

B

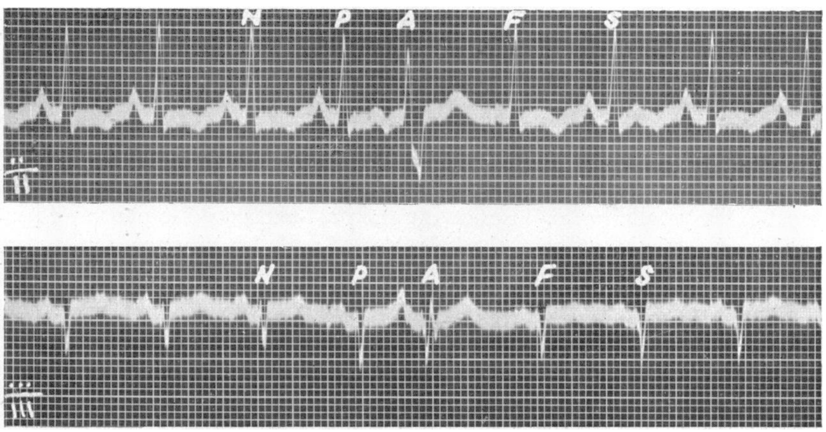



FIG. 4.—Auricular premature systoles (*APS*) showing increased duration of both QRS and RS–T—two examples. N, normal beat; P, beat preceding the *APS*; A, *APS*; F, beat following the *APS*; S, subsequent beat. Measurements in seconds.

(A)

| | QRS | RS–T | Q–T |
|---|---|---|---|
| N | 0·08 | 0·24 | 0·32 |
| P | 0·085 | × | × |
| A | 0·12 | 0·32 | 0·44 |
| F | 0·08 | 0·26 | 0·34 |
| S | 0·08 | 0·25 | 0·33 |

(B)

| | QRS | RS–T | Q–T |
|---|---|---|---|
| N | 0·06 | 0·225 | 0·285 |
| P | 0·055 | × | × |
| A | 0·10 | 0·26 | 0·36 |
| F | 0·06 | 0·23 | 0·29 |
| S | 0·07 | 0·22 | 0·29 |

*Comparison of* APS *with the following beat.* The beat following an *APS* has a prolonged RS–T interval. When we compared the post-extrasystolic beat with the *APS*, the RS–T prolongation of the former was a practically constant finding. But even when we compared the post-extrasystolic beat with the normal beat N, prolongation of RS–T was found in a high percentage of cases (Table III). The prolongation usually amounted to 0·01 to 0·02 sec., but greater prolongation, of 0·06, 0·07, and even 0·08 sec. occurred occasionally. The effect of the pause was obvious. In general, the longer the pause after an *APS*, the greater was the prolongation of the RS–T interval of the following beat; but this relationship was by no means consistent, and actual shortening of the RS–T interval of the following beat occasionally occurred, even after a long pause.

TABLE III

COMPARISON OF POST-EXTRASYSTOLIC BEAT (F) WITH AURICULAR PREMATURE SYSTOLE (*APS*) AND WITH NORMAL BEAT (N)

| | RS–T longer (per cent) | RS–T identical (per cent) | RS–T shorter (per cent) |
|---|---|---|---|
| F compared with *APS* .. | 97 | 1 | 2 |
| F compared with N .. .. | 68 | 22 | 10 |

## COMMENT

Our investigation has established two facts: The RS–T interval (and thereby the Q–T interval) of an auricular premature systole is shortened, that of the post-extrasystolic beat is lengthened. Shortening of the electrical systole is a feature of auricular premature systoles not heretofore known, and its significance is not yet understood. Observations on the Q–T interval of normal beats were published by us (Berliner, 1931). Since then, knowledge of this subject has not materially increased. Varied factors, e.g. blood calcium level and digitalis, are known to influence the Q–T interval of normal beats but the determining factor unquestionably is the heart rate. The Q–T interval is shorter for faster rates and longer for slower rates. The relationship of heart rate and Q–T interval is expressed by Bazett's formula (1920)

$$\text{Q–T}=\text{K}\cdot\sqrt{\text{cycle length}},$$

the constant K being 0·37 for men and 0·40 for women. This formula or any of its variants obviously applies to beats occurring in regular rhythm and cannot be used to predict the Q–T interval of premature beats. It was, therefore, not employed in the present study. Rate or cycle length may, however, still be found to have an influence on the Q–T interval of premature beats and post-extrasystolic beats. Following the post-extrasystolic pause we found the Q–T interval lengthened. There, at least, the influence of cycle length appears very probable.

We were unable to find out what causes the variations of RS–T duration discovered by us. We may, however, have a clue in the contrast between shortened RS–T in the *APS* and lengthened RS–T in the post-extrasystolic beats. In a separate investigation of ventricular premature systoles, which will be reported later, we found exactly the same conditions; the RS–T intervals of ventricular premature beats are also shorter than those of normal beats (irrespective of the large size of the T waves), and the RS–T intervals of the post-extrasystolic beats are longer. It seems most likely to us that the shortening of RS–T in all premature systoles is caused by their prematurity. Prematurity results in incomplete diastolic filling of the ventricles, and this may be reflected in a shorter duration of RS–T. The post-extrasystolic pause, on the other hand, leads to greater diastolic filling of the ventricles, and this may give rise to an increased RS–T duration. Should this assumption be proved correct, we might be brought nearer to the solution of a basic electrocardiographic problem. The significance of RS–T interval variations in *normal* beats has always been obscure. It might then be found that they too are caused by changes in the diastolic filling of the ventricles.

In conclusion, a remark about measurements of Q–T intervals in general may be in order. Our findings clearly show that the QRS and RS–T intervals vary separately and perhaps independently. They should, therefore, be studied separately. Albers and Bedbur (1941) did that and found that the duration of QRS depends on the heart rate, the duration of RS–T on the heart rate, the age of the patient, and the degree of axis deviation. It was mentioned before that we repeatedly found prolongation of QRS associated with marked shortening of RS–T; the resulting Q–T interval was normal or nearly normal (Fig. 3). Simple measurements of Q–T in such cases would not have revealed the marked shortening of RS–T. Our experience, therefore, convinces us that measurements of Q–T alone are insufficient and should be replaced by separate measurements of QRS and RS–T.

## Summary and Conclusions

The duration of QRS and RS–T of 100 auricular premature systoles (*APS*) was measured and two facts were established: (1) the RS–T interval of an *APS* is usually shorter than that of normal beats, and (2) the RS–T interval of the post-extrasystolic beat is lengthened.

The greatest degrees of shortening of RS–T were found in *APS* with marked widening of their QRS complexes. As a result, the Q–T interval of these *APS* was no longer than that of the normal beats.

QRS and RS–T intervals of *APS* vary independently and should be studied separately. Simple measurements of Q–T intervals are insufficient.

Shortening of RS–T in *APS* is probably caused by the incomplete diastolic filling of the ventricles resulting from prematurity, while lengthening of RS–T in post-extrasystolic beats is probably caused by greater diastolic filling.

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